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# Anticoagulant Rodenticides and Wildlife: Introduction

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# Chapter 1

## Anticoagulant Rodenticides and Wildlife:

### Introduction

Nico W. van den Brink, John E. Elliott, Richard F. Shore,  
and Barnett A. Rattner

## 1 Setting the Scene

Rodents began to associate with humans at least from the early Neolithic era with the beginnings of systematic sequestering of food stores by humans (Cucchi and Vigne 2006; Reperant and Osterhaus 2014). About the year 541, the Justinian plague started amid the central granaries and crowded, unsanitary conditions of the later Roman cities. The resulting pandemic was the first documented example of the potentially devastating impact of commensal rodents on European society. The primary reservoir host and source of the plague was the black rat (*Rattus rattus*), which has widely thought to have disseminated from South-East Asia along land and marine trade routes (McCormick 2003). That plague spread through late Roman and early medieval Europe until the eighth century (McCormick 2003). About 600 years later, the Black Death was also vectored by *R. rattus*. Both pandemics were caused by *Yersinia pestis*, possibly infecting *R. rattus* via endemically infected burrowing rodents along the trade routes of Central Asia (Reperant and Osterhaus 2014); however, the strains were different between the pandemics, and the occurrences seemed to be independent (Wagner et al. 2014). *R. rattus* is considered to be the commensal reservoir of *Y. pestis* and fleas, the vectors between rats and humans, although in

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some Nordic countries human-to-human infections are thought also to have been important (Hufthammer and Walløe 2013). Additionally, recent assessments based on analysis of climate data suggest that *R. rattus* did not provide a reservoir and that overland routes were not the pathway, rather *Yersinia pestis* was repeatedly introduced into Europe from Asia via maritime routes (Schmid et al. 2015). Currently, the human plague is still affecting Asian and African countries, with thousands of annual casualties (Butler 2009). Rodents also carry a wide range of other bacteria as well as ecto- and endoparasites and viruses that pose a potential risk to human health (Battersby 2015).

Rodents also affect human food resources, both by consumption and fouling. In East Africa, average total crop losses due to rodents, are estimated to sometimes reach 80–100% (Makundi and Massawe 2011). In Tanzania, the average yield loss of maize is estimated to be 5–15%, potentially feeding 2.3 million people and costing approximately \$40 million (Leirs 2003; 1998 United Nations data). In a study on crop damage due to rodents in Laos, it was shown that in certain regions losses may be up to 80% (Douangboupouha et al. 2003). There are reports that in European and American countries, rodents also may affect agricultural practices by fouling and feeding on animal feed and crops, and potentially by acting as reservoirs for veterinary diseases (Stenseth et al. 2003). Losses to stored food from consumption, spillage, damage and contamination are of the order of 1–10% (Smith and Meyer 2015), but rodents also degrade crop yields and cause structural damage.

When introduced to islands, alien rodent species can spread rapidly, and may have devastating effects on local biota. For instance, over 100 of 123 major island groups in the world have been colonised by *Rattus* species, preying on local fauna and consuming vegetation, often impeding regeneration of seedlings (Amori and Clout 2003; Howald et al. 1999). Besides commensal rodents, also other (small) mammalian species have been introduced to islands (e.g., possums and mustelids in New Zealand; Alterio and Moller 2000; Eason et al. 2010). Introduction by humans is often non-intentional, but eradication is extremely difficult, usually associated with high costs and environmental impact.

In more recent time, additional problems have been associated with commensal rodents. For instance, rodents can destroy power and ICT cables, and damage insulation and other building materials (Shumake et al. 2000). This not only results in direct costs to the infrastructure, but may also impede work and result in dangerous situations when people are relying on sensitive life-saving equipment (e.g., in hospitals). Overall, rat-mediated losses in the US (excluding any human health costs) could amount to \$19,000,000,000 per year (Pimentel et al. 2005), while estimates of equivalent costs for the UK are £200,000,000 per year (Battersby 2004).

The aforementioned socio/economic impacts of rodents clearly provide examples of the need to control commensal rodent populations. Historically, different methods have been applied, although we are unaware of any “scientific” descriptions of historic methods used. In ancient Egypt, Rome and India, cats (*Felis sp.*) and ferrets (*Mustela sp.*) have been associated with minimizing commensal rodent populations in agriculture, near granaries and food stores (Baldwin 1975; Faure and Kitchener 2009; Mark 2012). The painting “*Mérode Altarpiece*” by Robert Campin

(circa 1425–1428, Metropolitan Museum of Art, New York) shows a mousetrap, and although depiction of the mousetrap had a religious connotation and symbolism, it indicates the use of traps for rodent control in medieval times. The legend of the “Pied Piper of Hamelin” plays around 1284, and suggests that specific rat catchers were hired by people or cities to catch rats or to lure them away. In this period, primarily trapping was employed for rodent control although attempts were aimed at chemical control e.g. via herbs, to deter rodents. For instance John Gerard describes in the book “The Herball or Generall Historie of Plantes” (Gerard, 1597) how White Hellebore (*Veratrum album*) is effective in killing rodents: “The root giuen to drinke in the weight of two pence, taketh away the fits of agues, killeth mice and rats, being made vp with honey and floure or wheat” (sic). The roots of this plant species contain veratridine, a neurotoxic compound, a sodium ion channel agonist (Segura-Aguilara and Kostrzewa 2004).

The use of chemicals to control rodents (i.e. rodenticides) has been in practice for nearly a century, and is commonplace today. Most currently used rodenticides are anticoagulants, which prevent blood from clotting (Rattner et al. 2014). In the 1920s the discovery of the so called “Sweet clover disease”, causing haemorrhage and even mortality in cattle due to poorly stored clover hay, lead to the detection of dicoumarol as the causing compound, and to the further development of the first-generation anticoagulant rodenticides (FGARs) based on related to the structure of warfarin (Link 1959).

Schein (1950) described the effectiveness of W.A.R.F. 42 (warfarin) as an anticoagulant rodenticide, and made the remark that it was highly effective on a short term, but that “environmental changes, such as reduction in the harborage or food supply, would give more profound and more permanent control, and is therefore to be preferred”. In another trial, warfarin was also considered to be very effective in controlling rats (Wayland and Gaines 1950). In that study, they used a very simple bait-station in order to prevent children from accessing and consuming poison, a first attempt to minimise non-target exposure. In 1952, the Association of Schools of Public Health published a recommendation on the use of warfarin, stating its effectiveness and the “relatively small hazard to man and useful animals compared to the hazard offered by most other effective rodenticides” (Anonymous 1952). None of those early reports mention issues with environmental risks. In the US, the use of rodenticides was regulated through the Federal Insecticide, Fungicide, and Rodenticide Act of 1947, in which environmental risks were not included (Ward 1965). At that time, the use of prolin, a warfarin based AR, was also promoted as an alternative for other methods of rodent control (e.g., arsenic trioxide, barium carbonate, endrin, fluoroacetamide 1081, strychnine, thallium sulfate, zinc phosphide) because those “methods have shown varying degrees of success, but with questionable safety to wildlife populations” (Libby and Abrams 1966). This suggests that the use of ARs was seen to be an environmentally friendly alternative. However, it was around that time that development of resistance of rodents to warfarin and diphacinone was first reported (Boyle 1960; Cuthbert 1963; Lund 1964). Although resistance to anticoagulants in rodents was widespread by the early 1970s, and likely to continue to spread, the UK Ministry of Agriculture, Fisheries and Food

stated “the appearance of strains of *R. norvegicus* and *Mus musculus* resistant to anticoagulants in Britain, Denmark, Holland and the US is a matter of concern, but there is no reason why other countries should not continue to develop and use anticoagulant rodenticides wherever they are suitable” (Bentley 1972). It was also stated that birds are generally resistant to anticoagulants, without further reference or data.

Despite the prevailing opinion that warfarin was both safe and effective, evidence began to accrue that some rodent populations were developing resistance to some FGARs (e.g., warfarin and diphacinone, Boyle 1960; Cuthbert 1963; Lund 1964). Possible mechanisms of resistance development include mutations at the receptor site with decreased binding affinity of the compounds (Lasseur et al. 2006; Meerburg et al. 2014; Pelz and Prescott 2015) or by modulation of metabolic activity (Ishizuka et al. 2008; Markussen et al. 2007).

Putatively in response to the development of resistance in some populations, more acutely toxic ARs were developed and promoted, so called second-generation anticoagulant rodenticides (SGARs). These “super warfarins” were largely effective in controlling rats that had developed warfarin resistance; a single feed delivered a toxic dose, thereby increasing the efficacy of control measures and reducing the likelihood of resistance development. However, in recent years, resistance towards some SGARs has been reported in rat populations (Buckle 2013; Meerburg et al. 2014). Besides the issue of resistance in decreasing the efficacy of the use of ARs, the potential risks of rodenticides to non-target rodents and secondary poisoning of predators is now recognized. Due to their persistence, SGARs in particular have been reported to accumulate in non-target rodent and bird species directly feeding on the baits (Brakes and Smith 2005; Hoare and Hare 2006; Tosh et al. 2011; Sánchez-Barbudo et al. 2012), even impacting local population densities (Brakes and Smith 2005). Secondary exposure and some poisoning of predators is also widely reported (e.g., Newton et al. 1990; Shore et al. 1996; Berny et al. 1997; Stone et al. 1999; Alterio and Moller 2000; Fournier-Chambrillon et al. 2004; Walker et al. 2008; Sage et al. 2010; Murray 2011; Thomas et al. 2011; Christensen et al. 2012; Sánchez-Barbudo et al. 2012; Elliott et al. 2013), as well as to scavengers (Howald et al. 1999).

## 2 Regulation of ARs: Environmental Risks Versus Societal Needs

Anticoagulant rodenticides are regulated under different frameworks. For instance, in Europe they are regulated either as plant protection product or as biocide, depending on their use. As a plant protection product, ARs are used in some countries to protect crops on fields. Biocidal use focuses on the control of rodents around buildings, properties, and industrial sites, and authorization is granted on the need to protect human health. Agricultural, rural and urban use of ARs

regulated under the Federal Insecticide, Fungicide and Rodenticide Act in the U.S. and through the Pest Control Products Act by Health Canada, with recent legislation limiting use of SGARs. This is because SGARs are considered to be PBT-compounds (Persistent, Bioaccumulative, Toxic), and generally fail the thresholds set for environmental risks. However, due to the societal need to control rodents, and the current lack of alternatives, the use of SGARs is still permitted in many situations under strict regulation of application. It has been shown that the way ARs are used can modulate the risks of secondary poisoning of predators (Shore et al. 2006), and the development and application of best practices may decrease the risks of exposure of non-target species to ARs (Tosh et al. 2011). This would shift the regulation of ARs from risk assessment to risk management, which would require clear insights into the spatio-temporal risks that ARs may pose to non-target species. Currently, alternative approaches and methods are being developed, in order to minimise risks. Effectiveness of such approaches in reducing environmental risks to non-targets and preventing the development of resistance, however, is yet to be established.

Recently we presented and discussed additional mitigation options available to stakeholders (industry, pesticide applicators, regulators and the public) on this issue (Elliott et al. 2016). Among the approaches discussed were the broader adoption of integrated pest management (IPM) programs by industry, and particularly, the U.S. EPA's advocacy for expanded IPM, which is also the direction the EU is promoting for Europe, including quantifying the effectiveness of the IPM measures in mitigating risks. Furthermore, industry's response with the "Go Green" programs of large retail establishments is considered a very interesting way forward. The UK has taken leadership by developing an industry led and sponsored AR stewardship scheme. It involves development of best practice for usage, education and outreach programs targeted at key user sectors, and monitoring of the outcomes of stewardship on user practice and on levels of non-target exposure and impacts (CRRU 2017). The state of California in the US has applied the approach of Ecofees, which are collected on sales of ARs and other vertebrate control chemicals (Timm et al. 2004; Hornbaker et al. 2012). The revenues of such Ecofees are used for research on the toxicity and environmental effects of current use rodenticides, and of new alternative products and safe use practices.

The development of alternative approaches for the control of commensal rodents requires research on mechanisms of exposure and effects in target and non-target species under real application conditions. Quantification of the risks associated with current use rodenticides is essential for assessing the effectiveness of the alternatives in mitigating risk. This book aims to provide a state-of-the-art overview of the scientific advancements in the assessment of exposure, effects and risks that currently used ARs may pose to non-target organisms in the environment, along with practical guidance for characterization of hazards. This will be discussed in relation to their efficacy, and the societal needs for rodent control, and discussion of risk mitigation and development of alternatives.

### 3 Background, Rationale and Outline for the Book

The idea for this book grew out of two scientific sessions on the impact of anticoagulant rodenticides on non-target wildlife, held at annual meetings of the Society of Environmental Toxicology and Chemistry (SETAC); the first from May 20 to 24, 2012 in Berlin, Germany, and the second from November 11 to 15, 2012 in Long Beach, California, U.S.A. Impetus partly came from the widespread interest in the topic evident by coverage of the Long Beach session in the international science media (e.g., Nature; Lovett 2012) and covered further in other e-media such as Scientific American News). Furthermore, based on a growing body of scientific evidence gathered over the last 20 years, it is now recognised that large-scale use of anticoagulants may pose a global risk to vertebrate wildlife as acknowledged by international agencies and conventions (e.g., United Nations Environmental Convention on Migratory Species 2014). We perceived, therefore, a need for a volume that focused on the environmental impact of ARs on non-target wildlife *per se*, rather than more general information on the chemistry and toxicology of these pesticides such as in another recent book (Buckle and Smith 2015). This book is the first attempt to comprehensively bring together all the available information on the environmental risks associated with rodent control using ARs. The overall aim of the book is to highlight current state of knowledge which will: (i) help shape and identify mitigation methods and effort in such a way as to reduce risk, and (ii) identify key gaps and uncertainties in our understanding and thereby point to major areas of future research and regulatory need.

The book begins with an overview of anticoagulant rodenticide use around the world (Chap. 2). In the following chapters, the focus is on controlled laboratory studies of anticoagulant toxicity (Chap. 3) and their pharmacokinetics in target and non-target species (Chap. 4). Chapter 5 presents diagnostics and clinical signs of AR toxicity. The perspective then widens to first assess the causes, scale and effects of primary exposure in non-target species (Chap. 6), followed by a chapter on secondary exposure of predators (Chap. 7). The spatial dimensions of how exposure and impacts vary are considered in detail in Chap. 8, while Chap. 9 addresses the key ecological factors affecting AR uptake. The important topic of resistance in target populations is examined in Chap. 10 and finally, regulatory aspects (Chap. 11), risk mitigation (Chap. 12) and development of alternative rodent control methods (Chap. 13) are analyzed. The final chapter describes projected needs for rodent control in the future, discusses the sustainability of AR use, and the need for alternative non-chemical or new chemical methods that are effective while minimizing non-target risks.

All chapters of this book describe the scientific background of the different topics addressed, but also reach out to a wider audience. Because of the diversity of topics, it was intended to make each chapter accessible on its own, so authors were encouraged to introduce the chapters in depth. The reader is of course encouraged to read the full book, but in this way it is also possible to focus on specific topics of interest. We hope that this facilitates the reader to use the book as a base of information for discussions and possibly even decisions, as it was intended for.



## References

- Alterio N, Moller H (2000) Secondary poisoning of stoats (*Mustela erminea*) in a south island podocarp forest, New Zealand: implications for conservation. *Wildl Res* 27:501–508
- Amori G, Clout M (2003) Rodents on islands: a conservation challenge. In: Singleton G, Hinds L, Krebs C, Spratt D (eds) *Rats, mice and people: rodent biology and management*. Australian Centre for International Agricultural Research, Canberra, pp 62–68
- Anonymous (1952) Insecticides and rodenticides–1952 recommendations for use. *Public Health Rep* 67:455–458
- Baldwin JA (1975) Notes and speculation on the domestication of the cat in Egypt. *Anthropos* 70:428–448
- Battersby SA (2004) Public health policy – can there be an economic imperative? an examination of one such issue. *J Environ Health Res* 3:1–13
- Battersby SA (2015) Rodents as carriers of disease. In: Buckle A, Smith RH (eds) *Rodent pests and control*, 2nd edn. CAB International, Wallingford, pp 81–100
- Bentley EW (1972) Review of anticoagulant rodenticides in current use. *Bull World Health Organ* 47:275–280
- Berny PJ, Buronfosse T, Buronfosse F, Lamarque F, Lorgue G (1997) Field evidence of secondary poisoning of foxes (*Vulpes vulpes*) and buzzards (*Buteo buteo*) by bromadiolone, a 4-year survey. *Chemosphere* 35:1817–1829
- Boyle CM (1960) Case of apparent resistance of *Rattus norvegicus* Berkenhout to anticoagulant poisons. *Nature* 188:517–517
- Brakes CR, Smith RH (2005) Exposure of non-target small mammals to rodenticides: short-term effects, recovery and implications for secondary poisoning. *J Appl Ecol* 42:118–128
- Buckle A (2013) Anticoagulant resistance in the United Kingdom and a new guideline for the management of resistant infestations of Norway rats (*Rattus norvegicus* Berk.) *Pest Manag Sci* 69:334–341
- Buckle A, Smith R (2015) *Rodent pests and their control*. CABI, Wallingford
- Butler T (2009) Plague into the 21st century. *Clin Infect Dis* 49:736–742
- Christensen TK, Lassen P, Elmeros M (2012) High exposure rates of anticoagulant rodenticides in predatory bird species in intensively managed landscapes in Denmark. *Arch Environ Contam Toxicol* 63:437–444
- CRRU (2017) Think Wildlife: <http://www.thinkwildlife.org/stewardship-regime/>. Accessed 21 Feb 2017
- Cucci T, Vigne J-D (2006) Origin and diffusion of the house mouse in the Mediterranean. *Hum Evol* 21:95–106
- Cuthbert JH (1963) Further evidence of resistance to warfarin in the rat. *Nature* 198:807–808
- Douangboupouha B, Aplin KP, Singleton GR (2003) Rodent outbreaks in the uplands of Laos: analysis of historical patterns and the identity of nuu khii. In: Singleton G, Hinds L, Krebs C, Spratt D (eds) *Rats, mice and people: rodent biology and management*. Australian Centre for International Agricultural Research, Canberra, pp 103–111
- Eason C, Henderson R, Hix S, MacMorran D, Miller A, Murphy E, Ross J, Ogilvie S (2010) Alternatives to brodifacoum and 1080 for possum and rodent control-how and why? *New Zealand J Zool* 37:175–183
- Elliott JE, Hindmarch S, Albert CA, Emery J, Mineau P, Maisonneuve F (2013) Exposure pathways of anticoagulant rodenticides to nontarget wildlife. *Environ Monit Assess*:1–12
- Elliott JE, Rattner BA, Shore RF, Van Den Brink NW (2016) Paying the pipers: mitigating the impact of anticoagulant rodenticides on predators and savengers. *Bioscience* 66:401–407
- Faure E, Kitchener AC (2009) An archaeological and historical review of the relationships between felids and people. *Anthrozoös* 22:221–238
- Fournier-Chambrillon C, Berny PJ, Coiffier O, Barbedienne P, Dasse B, Delas G, Galineau H, Mazet A, Pouzenc P, Rosoux R, Fournier P (2004) Evidence of secondary poisoning of



- free-ranging riparian mustelids by anticoagulant rodenticides in France: implications for conservation of European mink (*Mustela lutreola*). *J Wildl Dis* 40:688–695
- Gerard, John (1597). *The Herball or Generall Historie of Plantes* (1st ed.). London: John Norton. Website: <https://archive.org/details/mobot31753000817749> (retrieved 31-07-2017)
- Hoare JM, Hare KM (2006) The impact of brodifacoum on non-target wildlife: gaps in knowledge. *N Z J Ecol* 30:157–167
- Hornbaker VL, Baldwin RA, Richards SN (2012) Potential fiscal impact of the rodenticide risk mitigation decision to the California Department of Food and Agriculture's rodenticide research program. In *Proceedings of the 25th Vertebrate Pest Conference* pp 164–168
- Howald GR, Mineau P, Elliott JE, Cheng KM (1999) Brodifacoum poisoning of avian scavengers during rat control on a seabird colony. *Ecotoxicology* 8:431–447
- Hufthammer AK, Walløe L (2013) Rats cannot have been intermediate hosts for *Yersinia pestis* during medieval plague epidemics in Northern Europe. *J Archaeol Sci* 40:1752–1759
- Ishizuka M, Tanikawa T, Tanaka KD, Heewon M, Okajima F, Sakamoto KQ, Fujita S (2008) Pesticide resistance in wild mammals – mechanisms of anticoagulant resistance in wild rodents. *J Toxicol Sci* 33:283–291
- Lasseur R, Longin-Sauvageon C, Videmann B, Billeret M, Berny P, Benoit E (2006) Warfarin resistance in a French strain of rats. *J Biochem Mol Toxicol* 19:379–385
- Leirs H (2003) Management of rodents in crops: the pied piper and his orchestra. In: Singleton G, Hinds L, Krebs C, Spratt D (eds) *Rats, mice and people: rodent biology and management*. Australian Centre for International Agricultural Research, Canberra, pp 183–190
- Libby JL, Abrams JI (1966) Anticoagulant rodenticide in paper tubes for control of meadow mice. *J Wildl Manag* 30:512–518
- Link KP (1959) The discovery of dicumarol and its sequels. *Circulation* 19:97–107
- Lovett RA (2012) Killing rats is killing birds. *Nature*. doi:10.1038/nature.2012.11824
- Lund M (1964) Resistance to warfarin in common rat. *Nature* 203:778
- Makundi RH, Massawe AW (2011) Ecologically based rodent management in Africa: potential and challenges. *Wildl Res* 38:588–595
- Mark JJ (2012) Cats in the ancient world. *Ancient History Encyclopedia*. <http://www.ancient.eu/article/466/>. Accessed 15 Feb 2017
- Markussen MD, Heiberg AC, Alsbo C, Nielsen PS, Kauppinen S, Kristensen M (2007) Involvement of hepatic xenobiotic related genes in bromadiolone resistance in wild Norway rats, *Rattus norvegicus* (Berk.) *Pestic Biochem Physiol* 88:284–295
- McCormick M (2003) Rats, communications, and plague: toward an ecological history. *J Interdiscip Hist* 34:1–25
- Meerburg BG, van Gent-Pelzer MPE, Schoelitsz B, van der Lee TAJ (2014) Distribution of anticoagulant rodenticide resistance in *Rattus norvegicus* in the Netherlands according to Vkorc1 mutations. *Pest Manag Sci* 70:1761–1766
- Murray M (2011) Anticoagulant rodenticide exposure and toxicosis in species of birds of prey presented to a wildlife clinic in Massachusetts, 2006–2010. *J Zoo Wildl Med* 42:88–97
- Newton I, Wyllie I, Freestone P (1990) Rodenticides in British barn owls. *Environ Pollut* 68:101–118
- Pelz HJ, Prescott CV (2015) Resistance to anticoagulant rodenticides. In: Buckle AR, Smith RH (eds) *Rodent pests and their control*, 2nd edn. CAB International, Wallingford, pp 101–122
- Pimentel D, Zuniga R, Morrison D (2005) Update on the environmental and economic costs associated with alien-invasive species in the United States. *Ecol Econ* 52:273–288
- Rattner BA, Lazarus RS, Elliott JE, Shore RF, van den Brink N (2014) Adverse outcome pathway and risks of anticoagulant rodenticides to predatory wildlife. *Environ Sci Technol* 48:8433–8445
- Reperant LA, Osterhaus AD (2014) The human-animal interface. In: Atlas RM, Maloy S (eds) *One Health people, animals, and the environment*. ASM Press, Washington, DC, pp 33–52
- Sage M, Fourel I, Coeurdassier M, Barrat J, Berny P, Giraudoux P (2010) Determination of bromadiolone residues in fox faeces by LC/ESI-MS in relationship with toxicological data and clinical signs after repeated exposure. *Environ Res* 110:664–674
- Sánchez-Barbudo IS, Camarero PR, Mateo R (2012) Primary and secondary poisoning by anticoagulant rodenticides of non-target animals in Spain. *Sci Total Environ* 420:280–288

- Schein MW (1950) Field test of the efficiency of the rodenticide compound W.A.R.F. 42. Public Health Rep (1896–1970) 65:368–372
- Schmid BV, Büntgen U, Easterday WR, Ginzler C, Walløe L, Bramanti B, Stenseth NC (2015) Climate-driven introduction of the Black Death and successive plague reintroductions into Europe. Proc Natl Acad Sci 112:3020–3025
- Segura-Aguilara J, Kostrzewa RM (2004) Neurotoxins and neurotoxic species implicated in neurodegeneration. Neurotox Res 6:615–630
- Shore RF, Birks JDS, Freestone P, Kitchener AC (1996) Second-generation rodenticides and polecats (*Mustela putorius*) in Britain. Environ Pollut 91:279–282
- Shore RF, Malcolm HM, McLennan D, Turk A, Walker LA, Wienburg CL, Burn AJ (2006) Did foot-and-mouth disease-control operations affect rodenticide exposure in raptors? J Wildl Manag 70:588–593
- Shumake SA, Sterner RT, Gaddis SE (2000) Repellents to reduce cable gnawing by wild Norway rats. J Wildl Manag 64:1009–1013
- Smith RH, Meyer AN (2015) Rodent control methods: non-chemical and non-lethal chemical, with specific reference to food stores. In: Buckle AR, Smith RH (eds) Rodent pests and their control, 2nd edn. CAB International, Wallingford, pp 101–122
- Stenseth NC, Leirs H, Skonhøft A, Davis SA, Pech RP, Andreassen HP, Singleton GR, Lima M, Machang'u RS, Makundi RH, Zhang Z, Brown PR, Shi D, Wan X (2003) Mice, rats, and people: the bio-economics of agricultural rodent pests. Front Ecol Environ 1:367–375
- Stone WB, Okoniewski JC, Stedelin JR (1999) Poisoning of wildlife with anticoagulant rodenticides in New York. J Wildl Dis 35:187–193
- Thomas PJ, Mineau P, Shore RF, Champoux L, Martin PA, Wilson LK, Fitzgerald G, Elliott JE (2011) Second generation anticoagulant rodenticides in predatory birds: probabilistic characterisation of toxic liver concentrations and implications for predatory bird populations in Canada. Environ Int 37:914–920
- Timm RM, Schnabel DL, Salmon TP, Gorenzel WP, Dechoretz N, Meyers M (2004) California's rodenticide surcharge program: history and accomplishments. In: Proceedings of the 21st Vertebrate Pest Conference, pp 350–356
- Tosh DG, Shore RF, Jess S, Withers A, Bearhop S, Montgomery WI, McDonald RA (2011) User behaviour, best practice and the risks of non-target exposure associated with anticoagulant rodenticide use. J Environ Manag 92:1503–1508
- United Nations Convention on Migratory Species (2014) Review of the ecological effects of poisoning on migratory birds. [http://www.cms.int/sites/default/files/document/COP11\\_Inf\\_34\\_Review\\_effects\\_of\\_Poisoning\\_on\\_Migratory\\_Birds\\_Eonly.pdf](http://www.cms.int/sites/default/files/document/COP11_Inf_34_Review_effects_of_Poisoning_on_Migratory_Birds_Eonly.pdf). Accessed 21 Feb 2017
- Wagner DM, Klunk J, Harbeck M, Devault A, Waglechner N, Sahl JW, Enk J, Birdsell DN, Kuch M, Lumibao C, Poinar D, Pearson T, Fourment M, Golding B, Riehm JM, Earn DJD, DeWitte S, Rouillard J-M, Grupe G, Wiechmann I, Bliska JB, Keim PS, Scholz HC, Holmes EC, Poinar H (2014) *Yersinia pestis* and the plague of Justinian 541–543 AD: a genomic analysis. Lancet Infect Dis 14:319–326
- Walker LA, Turk A, Long SM, Wienburg CL, Best J, Shore RF (2008) Second generation anticoagulant rodenticides in tawny owls (*Strix aluco*) from Great Britain. Sci Total Environ 392:93–98
- Ward JC (1965) The functions of the Federal Insecticide, fungicide, and rodenticide act. Am J Public Health Nations Health 55:27–31
- Wayland JH, Gaines TB (1950) Control of Norway rats with residual rodenticide warfarin. Public Health Rep (1896–1970) 65:1537–1555